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The association of sleep disturbances with endocrine and perceived stress reactivity measures in male employees

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Abstract

Evidence on the relationship between stress reactivity and sleep is conflicting. This study examined the association between disturbed sleep and perceived and endocrine stress reactivity independently of age, body mass index (BMI), and chronic stress. One hundred and twenty middle-aged men were exposed to the Trier Social Stress Test for Groups. The Pittsburgh Sleep Quality Index and the Perceived Stress Reactivity Scale were used to assess sleep and perceived stress reactivity, respectively. Endocrine stress reactivity was examined by assessing salivary cortisol levels. Regression analyses showed that men with disturbed sleep had blunted overall cortisol responses (b = -18.246, p = .044) but the association did not survive adjustment for age, BMI, and chronic stress. In contrast, poor sleep was associated with heightened perceived stress reactivity independently of age and BMI (b = 0.235, p = .005), but additional adjustment for chronic stress attenuated the relationship and only chronic stress remained a significant predictor of perceived stress reactivity (b = 0.470, p < .001). Cortisol and perceived stress reactivity were uncorrelated. In summary, our study indicates associations between sleep disturbances and stress reactivity were not independent of BMI and chronic stress levels, and endocrine and perceived stress reactivity were dissociated.

Keywords: Sleep, cortisol, stress, stress reactivity, Trier Social Stress Test

The Association of Sleep Disturbances with Endocrine and Perceived Stress Reactivity Measures in Male Employees

Insufficient and disturbed sleep are linked with adverse health conditions including cardiovascular disease (Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011) obesity (Wu, Zhai, & Zhang, 2014), type 2 diabetes (Cappuccio, D'Elia, Strazzullo, & Miller, 2010) hypertension (Pepin et al., 2014), and depression (Salo et al., 2012). Sleep problems might contribute towards these adverse health effects by negatively influencing the stress system. Specifically, elevated (Chrousos, 2009) as well as attenuated responses of the stress system (Phillips, Ginty, & Hughes, 2013) can be maladaptive for health, and poor sleep is related to both heightened and attenuated endocrine stress responses (Minkel et al., 2014; Wright, Valdimarsdottir, Erblich, & Bovbjerg, 2007). However, acute stress responses are also influenced by other factors, for instance, physical exercise (e.g., Klaperski, von Dawans, Heinrichs, & Fuchs, 2014) and chronic stress (Matthews, Gump, & Owens, 2001), and both of these are also linked with sleep quality and vice versa (Åkerstedt et al., 2002; Booth et al., 2012; Swanson et al., 2011). Notably, this issue has been largely neglected in the existing literature.

The acute stress response constitutes reactions from the sympathetic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis, which this study will focus on. Evidence shows that sleep behavior is closely linked with the HPA axis as optimal sleep (in terms of duration and quality) is associated with a healthy diurnal profile of cortisol release as indicated, for example, by higher levels of cortisol in the morning, its lower concentration in the evening and a steeper slope of cortisol secretion (Meerlo, Sgoifo, & Suchecki, 2008). Relatedly, experimental and population-based research tested the plausible impact of sleep deprivation and fragmentation on basal levels of cortisol, an important biomarker of the HPA axis (Kirschbaum & Hellhammer, 2000), and numerous studies reported raised as well as blunted cortisol concentrations (e.g., Hansen et al., 2012; Reynolds et al., 2012; Stamatakis & Punjabi, 2010). As elevated cortisol levels have been found to be associated with conditions such as type 2 diabetes (Korenblum et al., 2005) and all-cause mortality (Kumari,

Shipley, Stafford, & Kivimaki, 2011), while blunted cortisol responses have been reported in clinical populations including patients with depression (Burke, Davis, Otte, & Mohr, 2005) or chronic fatigue (Scott, Medbak, & Dinan, 1998), sleep seems to play an important role with regard to basal cortisol levels and stress-related diseases.

In addition to these findings for basal cortisol levels, a recent review has further suggested that poor sleep, in particular if prolonged, may also interfere with acute endocrine stress responses (Phillips et al., 2013). However, there has been limited research on the role of insufficient sleep or its poor quality on cortisol reactivity and findings are conflicting. For instance, in one of the first studies of this kind conducted with 31 young adolescents greater self-reported sleep problems were found to be related to lower cortisol responses to the Trier Social Stress Test (Capaldi, Handwerger, Richardson, & Stroud, 2005). In contrast, Räikkönen et al. (2010) reported an association between low actigraphy-based sleep efficiency and increased cortisol reactivity to the Trier Social Stress Test for children in a sample of almost 300 8-year-olds. These data were corroborated by a recent investigation in 83 young adolescents where poor sleep quality was related to heightened cortisol responses to the Trier Social Stress Test (Mrug, Tyson, Turan, & Granger, 2016). Conflicting findings have emerged for adults as well: Wright and colleagues (2007) found that lower actigraphy-based sleep efficiency was associated with blunted cortisol responses to the Stroop colour-word interference stress task in 53 women; yet, objective sleep quantity and subjective sleep quality and quantity were not related to cortisol reactivity. In contrast, other studies found subjective poor sleep quality to be associated with greater cortisol reactivity to a cold-pressor task (Goodin, Smith, Quinn, King, & McGuire, 2012) and the Trier Social Stress Test in young adults (Bassett, Lupis, Gianferante, Rohleder, & Wolf, 2015). A recent experimental study by Minkel and colleagues (2014) corroborates these findings by showing that one night of total sleep deprivation was associated with an increased cortisol response to the Trier Social Stress Test, when compared with controls. Overall, a paucity of research and mixture of methods in this field hampers a better understanding of associations

between sleep quality and stress responses; especially findings for larger samples based on adult and male populations are still lacking.

Furthermore, it is important to consider that stress has been found to prospectively predict reduced sleep quality (Ota et al., 2009; Rugulies, Norborg, Sorensen, Knudsen, & Burr, 2009), and that negative life events and chronic stress have also been associated with altered stress responses including reduced cortisol reactivity (Carroll, Phillips, Ring, Der, & Hunt, 2005; Loft et al., 2007; Matthews et al., 2001). This raises the possibility that the relationship between sleep patterns and cortisol reactivity may not be independent of an individual's chronic stress level. However, only Wright et al. (2007) actually explored whether participants' perceived stress seven days prior to the stress task impacted the relationship between cortisol reactivity and sleep quality; they found no association with regard to objective sleep but did not report any results for subjective sleep. Apart from that, physiological and psychological or perceived stress reactivity have often been shown to be dissociated (Campbell & Ehlert, 2012; Klaperski, von Dawans, Heinrichs, & Fuchs, 2013); thus, it is important to also explore participants' perceived stress reactivity which has been shown to be positively associated with sleep disturbances and chronic stress (Schulz, Jansen, & Schlotz, 2005). This may improve understanding of the meaning of the endocrine stress response, especially with regard to inconsistent findings showing that elevated, blunted as well as absent responses to stress can be maladaptive to health (Kudielka, Hellhammer, & Wüst 2009).

Hence, the current study set out to investigate the important yet under-researched associations between sleep quality, stress responses and chronic stress by applying a standardized stressor (the Trier Social Stress Test for Groups, TSST-G) to a large male sample. The *first aim* of the study is to test whether sleep quality is associated with the physiological response to stress. In line with previous studies in adult populations we hypothesised that participants with poor sleep quality would have higher cortisol responses to the Trier Social Stress Test for Groups than those reporting good sleep. The *second aim* of our study is to better understand the relationship between subjective sleep, cortisol

reactivity and chronic stress. As chronic stress has been found to have an impact on stress reactivity we expected chronic stress to be related to cortisol reactivity and hypothesised that is might also influence the relationship between cortisol reactivity and subjective sleep quality. To further understand associations with perceived stress responses the *third aim* of the study is to test whether participants with greater sleep problems also report higher perceived stress reactivity.

Methods

Study Design

The research questions of this article are examined by analysing baseline data from a larger experimental study (for a more detailed description see Klaperski et al., 2014). The main study examined the stress buffer hypothesis and the cross stressor adaptation hypothesis by means of a randomized controlled trial (RCT). The present study only looks at the pre-intervention baseline data of the RCT, which included, amongst others, an endocrine stress response assessment and sleep, chronic stress, and perceived stress reactivity measures. Since the targeted number of subjects could not run through the study at once, and as a means of controlling for possible seasonal effects the study was conducted in two identical waves (wave 1: February to June 2012, n = 76; wave 2: August to December 2012, n = 73). The study had full ethical approval.

Participants

The target sample consisted of healthy male office workers who did not regularly engage in physical exercise and relaxation techniques, as these two activities constituted the intervention conditions (not described here). Male employees from several banking and insurance companies and civil service facilities located in southern Germany were asked to complete a screening survey. Of 474 men who participated in the screening survey, n = 228 participants were not regularly engaging in exercise and relaxation techniques, and out of these n = 149 men were eligible and invited to participate in the study (eligibility criteria were not made clear in advance). Exclusion criteria were: (1) severe acute or chronic medical illness, a current psychiatric disorder, or psychotherapy; (2) disorders or injuries preventing

them from participating in the programme; (3) substance abuse; (4) lack of fluency in German language. As the original study did not focus on investigating sleep participants were not explicitly asked whether they suffered from sleep-related disorders. A total of *n* = 142 subjects completed the baseline assessment which is of interest in the current study, but *n* = 5 participants withdrew their consent in the course of the study, resulting in a data sample of *n* = 137 at T1. As this study examines the cortisol stress response statistical analyses will furthermore be controlled for factors which are known to significantly impact the response of this stress marker (Phillips et al., 2013). Specifically, in correspondence with Klaperski at al. (2014), three subjects with a body mass index (BMI) over 35, nine subjects who smoked more than five cigarettes per day, one subject who used cortisone compounds, and one subject who drank alcohol before the assessment were excluded from the analyses. Furthermore, sleep data from three subjects were missing as they did not fully complete the sleep questionnaire, thus *n* = 120 men were included in the statistical analysis of the present study. See Figure 1 for a detailed participant flow chart.

Procedure

The baseline testing sessions took place approximately 4 weeks after the screening survey. Each session included a stress response assessment (approximately 2 h) and a fitness test (approximately 1 h), but since the latter is of no relevance for the present study it will not be described here. All testing sessions were conducted in groups of three men who were not acquainted with each other and took place between 3:00 p.m. and 9:00 p.m. To control for diurnal variations of cortisol release cortisol samples were taken between 3:30 p.m. and 8:00 p.m. A narrower or earlier window of cortisol collection was not feasible as all participants were to be tested within two weeks and as the sample consisted of office workers who were not available before the afternoon. Participants were requested to refrain from exhaustive physical exercise three days prior to the laboratory visit as well as from exercise, alcohol, coffee, and black and green tea 24 hours prior to the session. Participants were also advised not to smoke or eat 90 minutes prior to testing, and to have a regular breakfast and lunch but to refrain from juices, cola, and chewing gum on the day of the

testing session. Upon arriving at the research laboratory participants were given information about the study, were asked to provide written consent and to complete a health questionnaire.

Participants' stress responses were assessed by measuring individual cortisol responses to the Trier Social Stress Test for Groups. The Trier Social Stress Test for Groups is a standardized motivated performance task protocol (von Dawans, Kirschbaum, & Heinrichs, 2011) that combines high levels of uncontrollability and socio-evaluative threat. Subjects deliver an unrehearsed speech and complete a mental arithmetic task in front of a camera and two judges, which reliably and validly induces psychosocial stress (Dickerson & Kemeny, 2004). In the present study, the Trier Social Stress Test for Groups was adapted to groups of three instead of six persons. All subjects underwent a preparation period (45 min), a presentation period (14 min), and a resting period (60 min) (see Figure 2).

During preparation, the subjects completed initial questionnaires, saliva sampling was explained, the upcoming Trier Social Stress Test task was introduced, and participants were given 10 min to prepare for a job interview. At the end of the preparation period participants were led into the stress test presentation room where they stood in an upright standing position separated by dividing walls in front of a camera and a male as well as a female jury member. During the presentation period only the male member of the committee talked to the subjects, while the female member permanently observed the subjects; both judges were instructed to withhold any verbal and non-verbal feedback at all times. Each of the three participants was first given 3 min to present his speech (random order). Following all three speeches, the subjects completed an unannounced serial subtraction task, with each subject taking three 30 sec turns in alternation. After 14 min the committee thanked the subjects and left after the investigator had reentered the room. At this point the resting period started and the subjects stayed in their upright standing positions for 10 more minutes and filled out questionnaires. Afterwards the subjects were guided back to the first room, where they sat quietly and filled out questionnaires until saliva sampling was completed (for further details see Klaperski et al. [2014] and von Dawans et al. [2011]).

Measures

BMI, physical activity, and trait anxiety levels. Participants' weight and height were assessed at the beginning of the fitness test and used for BMI calculation. All questionnaires described in the present study were completed in the course of the laboratory testing session during the resting period after the Trier Social Stress Test for Groups. Apart from the main measures described below the present study provides information about physical exercise and trait anxiety levels as part of the sample description. Physical exercise levels were assessed with the Physical Activity and Sport Activity Questionnaire from Fuchs, Klaperski, Gerber, and Seelig, (2015). Trait anxiety was measured with the State Trait Anxiety Inventory (STAI; Laux, Glanzmann, Schaffner, & Spielberger, 1981). The internal consistency of the trait anxiety scale in this study was very good (Tavakol & Dennick, 2011) (Cronbach's α = .93).

Sleep quality. Participants completed the German version of the Pittsburgh Sleep Quality Index, which uses a retrospective 2 week time frame (PSQI; Riemann, Backhaus, & Schramm, 1996), as sleep measure. The Pittsburgh Sleep Quality Index consists of 19 single items which measure sleep quality along seven components: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction; the sum of these components yields a global sleep score (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Items were rated on a four-point scale (e.g. "During the last two weeks, how would you rate your sleep quality overall?") or specific values were indicated (e.g. "During the last two weeks, what time have you usually gone to bed at night?"). Participants were asked to base their answers on the majority of days and nights of the previous two weeks. The internal consistency of the overall Pittsburgh Sleep Quality Index scale was good with a Cronbach's α of .74. For the purpose of analyses presented here we computed a dichotomous grouping variable for sleep quality by means of a median-split. Accordingly, a "good sleep group" (overall sleep quality score ≤ 4; n = 80) and a "poor sleep group" (overall sleep quality score > 4; n = 40) emerged (see Statistical analysis section for further details about the sleep variable used in analyses

described here). We used a median split instead of a cut-off of > 5 (Buysse et al., 1989) since grouping sleep scores with this cut-off would have resulted in too few participants in the poor sleep category (*n* = 28).

Chronic stress. To measure chronic stress all subjects were given the short form of the Trier Inventory for Chronic Stress (TICS; Schulz, Schlotz, & Becker, 2004), a standardized, reliable and validated scale to measure chronic stress (Petrowski, Paul, Albani, & Brähler, 2012). The short form of the Trier Inventory for Chronic Stress contains 12 items, e.g.: “Although I do my best, my work is not appreciated”, “Times when I worry a lot and cannot stop”. Participants were asked to rate their answers on a five-point scale (from ‘never’ to ‘very often’) according to the experiences they had during the previous three months. Scores were averaged with greater scores reflecting higher chronic stress levels. The internal consistency of the TICS was good with a Cronbach’s α of .86.

Stress reactivity assessment. We measured salivary free cortisol levels to assess *cortisol reactivity* and the *overall cortisol response*. Cortisol release is a valid indicator of hypothalamic-pituitary-adrenal activity in response to an acute psychosocial stressor, especially when psychosocial stress is induced by a performance task containing social-evaluative threat and uncontrollability (Dickerson & Kemeny, 2004). The free, biologically active cortisol fraction in the blood can be reliably and validly assessed through the measurement of salivary free cortisol. Salivary free cortisol gradually increases within about 10 min, reaching its peak 10 to 30 min after stressor cessation (Foley & Kirschbaum, 2010). We collected six saliva samples from each participant before (-3 min relative to stressor onset) and after stress exposure (+15, +25, +40, +55, +75 min; see Figure 1) using a commercially available synthetic swab sampling device (Salivette® Blue cap; Sarstedt, Germany). Participants were asked to gently move the swab around in their mouth but not to chew for 1 min per sample. The saliva samples were stored at -20°C and sent to Dresden LabService GmbH (Germany) for biochemical analysis, where they were thawed and spun at 21°C at 3000 rpm for 3 min to obtain clear saliva; free cortisol concentrations (nmol/l) were determined by a luminescence immunoassay for the in-vitro-diagnostic quantitative

determination of cortisol in human saliva (IBL International). Interassay coefficients of variation were below 5%; all samples were suitable for analysis.

We used the Perceived Stress Reactivity Scale (PSRS; Schlotz, Yim, Zoccola, Jansen, & Schulz, 2011) to assess *perceived stress reactivity*. The scale consists of 29 items. Each item describes a potentially stressful situation (e.g., "When I am under stress") and provides three answer options representing a low (e.g., "I usually enjoy my leisure time"), a medium ("I usually have difficulty enjoying my leisure time"), and a high stress response ("I usually can't enjoy my leisure time at all"). The items constitute six subscales: reactivity to work overload, reactivity to social conflicts, reactivity to social stress, reactivity to failure, anticipatory reactivity, and prolonged reactivity. Usually, the overall perceived stress reactivity score is the average of all subscales, but in the present study, as to reduce participant burden, only the three subscales reactivity to social stress (5 items), anticipatory reactivity (4 items), and prolonged reactivity (4 items) were included and constituted the overall score. Greater scores indicated higher stress reactivity. The internal consistency of the Perceived Stress Reactivity Scale was good with a Cronbach's α of .70, .81, and .76 for the each of the subscales, respectively, and .77 for the overall value constituted by the three subscales. It is important to note perceived stress reactivity has not been assessed with regard to the Trier Social Stress Test for Groups in this study; it is a questionnaire-based subjective assessment of stress reactivity in general.

Statistical Analysis

Cortisol levels were calculated and analysed as follows: First, we natural logarithmized all cortisol values to normalize the positively skewed data. Second, we assessed whether the stressor indeed elicited a significant stress response as intended. For this purpose, we conducted a t-test in which the cortisol level directly before stress onset (sample 1) was compared with the cortisol level 25 min after stress onset (sample 3). Third, to assess the stress response we calculated the area under the individual response curve with respect to the ground (AUC_G) by using the formula described previously (Pruessner, Kirschbaum,

Meinlschmid, & Hellhammer, 2003)[1]. We calculated two different stress response measures: First, stress reactivity was computed in accordance with Klaperski et al. (2014) for the time period between the 1st and 3rd sample (sample before stress onset to sample 10 min after stressor cessation where cortisol peaked on average). We will refer to this measure as "Cortisol reactivity sample 1-3". Second, the overall cortisol response was calculated for the whole stress assessment time period between the 1st and the 6th sample; in the following we will refer to this measure as "Overall cortisol response". We chose to include this alternative cortisol calculation to enable a better comparison of our results with previous studies which used this approach (e.g., Miller et al., 2013).

In the analyses described here sleep was treated as a dichotomous variable as to enable a better comparison with past studies (e.g., Bassett et al., 2015; Wright et al., 2007) which followed a similar approach to data analysis. We also compared good and poor sleepers on a number of variables relevant to our study, as detailed in Table 1 below. Finally, preliminary analysis revealed that sleep scores on the Pittsburgh Sleep Quality Index were highly positively skewed (S = 1.49, Std. Error S = .22), a common finding (e.g., Bassett et al., 2015). As in this study of healthy men participants had on average good sleep quality (Md = 4.0; M = 4.35, SD = 2.63), a median split at > 4 (and not at > 5 which is the usual cut-off value) was used to differentiate between men with better and worse sleep quality.

T-tests were used to assess systematic differences between good and poor sleepers with regard to age, BMI, sleep quality, chronic stress levels, perceived stress reactivity, trait anxiety levels, physical exercise, and baseline cortisol levels. The relationship between sleep quality, chronic stress level, cortisol reactivity, overall cortisol response, perceived stress reactivity, BMI, and trait anxiety was further examined by means of a correlational analysis. The results are presented as means (SD) and p-values and Pearson correlation coefficients, as appropriate.

[1] The AUC_G is more suitable than repeated measurement analyses for analysing physiological data with numerous points of measurement and different time distances between measurements (Pruessner et al., 2003). We preferred the AUC_G to the AUC with respect to increase (AUC_I) as we were interested in the total amount of the response (Federenko et al., 2004) and as the AUC_I is based on the reference to the first value leading to a greater impact of the first value (Pruessner et al., 2003).

The associations between sleep quality and the *cortisol response* were tested with hierarchical linear regressions. Separate models were conducted for the two dependent variables *Cortisol reactivity sample 1-3* and *Overall cortisol response*. In model 1, only sleep quality was entered into the analysis, model 2 was adjusted for age and BMI, and in model 3 we additionally adjusted for chronic stress[2]. The association between sleep quality and *perceived stress reactivity* was analysed in the same fashion, with sleep being regressed on the stress measure (model 1), adjusting for age and BMI (model 2) and chronic stress (model 3). Preliminary screening for multicollinearity revealed that the correlation between chronic stress and perceived stress reactivity was $r = .63$ ($p < .01$), which does not exceed the $r = .80$ threshold considered problematic (Katz, 2011) (see Table 2). All regression results are presented as unstandardized regression coefficients (*b*), 95% confidence intervals (CI) and p-values (*p*). All data were analyzed with SPSS Statistics version 22.0.

Results

Participants' Characteristics

Participants' characteristics stratified by sleep quality are summarized in Table 1. Good and poor sleepers did not differ with respect to age, BMI, physical exercise and baseline cortisol levels. Significant differences were found with regard to overall sleep quality ($t(46.6) = -10.62$, $p < .001$, $r = .84$), chronic stress levels ($t(118) = -4.94$, $p < .001$, $r = .41$), perceived stress reactivity ($t(118) = -3.20$, $p = .002$, $r = .28$), and trait anxiety ($t(62) = -5.51$, $p < .001$, $r = .57$).

Cortisol Responses to the Stress Task

The analysis of cortisol responses revealed that the stress task indeed elicited a significant stress response as cortisol levels increased in all participants from cortisol sample 1 to sample 3 ($t(119) = -18.17$, $p < .001$, $r = .86$). Mean salivary cortisol levels (nmol/l) for

[2] Due to high correlations between trait anxiety and chronic stress and thus possible issue of multicollinearity, trait anxiety has not been included into the regression analyses as the focus of the study was to examine whether chronic stress would predict stress responses. However, it is evident that a relationship between trait anxiety and perceived stress reactivity exists (see Table 1).

good and poor sleep groups are presented in Figure 3. Mean perceived stress reactivity scores stratified by sleep quality are shown in Table 1

Correlation Analyses

Correlation analyses (see Table 2) showed that reduced sleep quality was associated with a *lower* overall cortisol response to the Trier Social Stress Test for Groups (yet, not with a lower cortisol reactivity) and a *higher* perceived stress reactivity. In line with the literature, lower sleep quality was more prevalent in those with higher chronic stress but was unrelated to BMI. High trait anxiety levels were similarly related to lower sleep quality. Perceived stress reactivity was linked to poor sleep and higher chronic stress levels; however, it was unrelated to both cortisol measures (see Table 2). Furthermore, participants with greater BMI had blunted overall cortisol responses to the stress task, but no such relationship was found for perceived stress reactivity.

Sleep and Stress Responses

Regression analyses (see Table 3) revealed that poor sleep quality was associated with a blunted overall cortisol response in the univariate model (b = -18.246, CI -35.98 to -0.51, p = .044; R^2 = 0.03). However, the relationship was no longer significant after adjusting for age and BMI in model 2 (R^2 = 0.09), or chronic stress in model 3 (R^2 = 0.09). The variable *Cortisol reactivity sample 1-3* was unrelated to sleep quality in all regression models (R^2 = 0.02, R^2 = 0.04, R^2 = 0.04 respectively). As depicted in Table 3 chronic stress was unrelated to both dependent cortisol variables. When sleep was entered into the regression as continuous instead of as dichotomous variable, the result pattern was identical with the exception of the overall cortisol response in Model 1, which became insignificant (b = -2.691, CI -5.91 to 0.53, p = .100; results not shown).

Examining perceived stress reactivity, regression analyses revealed that poorer sleep quality was predictive of greater perceived stress reactivity (see Table 3, model 1: b = 0.253, CI 0.10 to 0.41, p = .002; R^2 = 0.08). Results from model 2 (R^2 = 0.09) showed that the relationship was also independent of age and BMI (b = 0.235, CI 0.07 to 0.40, p = .005). However, the association did not remain significant after we adjusted for chronic stress in

model 3 (R^2 = 0.42); chronic stress became the only significant predictor of perceived stress reactivity (b = 0.470, CI 0.36 to 0.59, p < .001) and the variance the model explained significantly increased (p < 0.001). When sleep was entered into the regression as continuous instead of as dichotomous variable, the result pattern was identical (results not shown).

Discussion

This study was set out to investigate three aims: Firstly, to explore associations between sleep quality and the cortisol response by applying a standardized stressor, the Trier Social Stress Test for Groups, to a large sample of working men. Secondly, to test if the relationship between sleep quality and the cortisol response is independent of chronic stress levels. Thirdly, to explore the association between sleep quality, and chronic stress with perceived stress reactivity, and to relate this to the physiological stress response. With regard to the first aim our study revealed that poor sleep quality was not linked to an increased cortisol response but in contrast to a *blunted* one. However, this association was only found for one of two cortisol parameters and it was no longer significant after adjustment for BMI and age. The examination of the second aim revealed that the relationship between sleep quality and the cortisol response was unrelated to chronic stress; chronic stress was also not associated with cortisol reactivity or the overall cortisol response. With respect to the third aim of this study, our data showed that poorer sleep quality was significantly linked to heighten perceived stress reactivity, but the association was not independent of chronic stress which appeared to be a stronger predictor of perceived stress reactivity than sleep.

Sleep and the Cortisol Response

Previous investigations provided a very inconsistent picture of the association between disturbed sleep and the cortisol response to a stress task. In line with past studies with adult samples (e.g., Goodin et al., 2012; Minkel et al., 2014) we hypothesised that participants with poor sleep quality would show higher cortisol responses to the Trier Social Stress Test for Groups than participants with good sleep. However, our findings in unadjusted analyses

revealed the opposite result: Cortisol responses were blunted in participants with poor sleep, corroborating previous findings from a teenage sample by Capaldi et al. (2005). Notably, the association between subjective sleep and the overall cortisol response disappeared after adjustment for age and BMI, in turn supporting results from Wright et al. (2007), who found an association between cortisol reactivity and objective but not subjective sleep measures. However, the association with the overall cortisol response was only significant in the unadjusted Model 1 (see Table 3) when sleep scores were treated as dichotomous measure, and not as a continuously distributed variable. One plausible explanation for this pattern of findings may be that the association with the cortisol response is not a graded one, but is only apparent when potentially significant sleep disturbance is present.

There are various possible explanations for the discrepancy between our findings and previous studies which found different cortisol response patterns in poor sleepers: First, we used a large solely male adult, non-student sample which, to the best of our knowledge, has had not been examined yet. However, irregular work schedules or nigh-time work could mean that sleep patterns may subsequently mask cortisol diurnal secretory activity (e.g., Koller et al., 1994), and could also impact the cortisol response. This explanation is unlikely for our data as participants had regular work patterns. Second, in previous studies sleep quality has been assessed by means of many different methods and for different time periods. While our participants were asked to rate their sleep quality with regard to the last two weeks, Wright et al., (2007) assessed subjective sleep quality for only one night proceeding the testing session. Third, the relationship between sleep quality and the cortisol response may only be apparent when higher levels of sleep difficulties are present. In our study the mean Pittsburgh Sleep Quality Index score in the poor sleepers group was 7.2 (*SD* = 2.5). This suggests that global sleep quality was indeed impaired in this group. Nonetheless, only healthy participants were included in our study and we used a lower cut-off value than other studies; thus, it is plausible that only more severe sleep disturbance impact cortisol responses to stress. Finally, previous studies often failed to control for

confounding variables such as BMI (e.g., Capaldi et al., 2005; Goodin et al., 2012), which proved to be an important predictor of cortisol responses in our and previous analyses (Kudielka et al., 2009; Miller et al., 2013; Phillips et al., 2013). Indeed, when we repeated our models adjusting only for age but not for BMI we did find that men with disturbed sleep were significantly more likely to have blunted cortisol response to the Trier Social Stress Test for Groups (data not shown). This highlights that careful attention is needed when selecting confounding variables in studies on this topic.

Chronic Stress and the Sleep-Cortisol Response Relationship

In line with a previous study by Wright et al. (2007) but contrary to our expectations chronic stress was not associated with the cortisol response, and it also did not alter the relationship between sleep quality and the cortisol response. It seems plausible that we found no significant effects with regard to the sleep-cortisol response relationship because sleep itself did not have any predictive value after adjusting for BMI and age in the second regression model. However, this does not yet explain why chronic stress, cortisol reactivity and the overall cortisol response proved to be unrelated in the correlational analysis. This result is surprising as studies have found that chronic stress can influence individual's cortisol responses (Matthews et al., 2001). Moreover, in line with the literature, we found higher chronic stress levels to be correlated with BMI values (r = .20) as well as poor sleep (r = .41) (Åkerstedt, 2006; Torres & Nowson, 2007). Based on our findings we therefore still recommend to assess chronic stress levels and to control for this factor in studies relating sleep and cortisol, or more broadly stress reactivity, as the sparse existing evidence needs further replication before it can be concluded that the link between sleep patterns and stress responses is independent of chronic stress levels.

Sleep and Perceived Stress Reactivity

According to our hypothesis and in line with findings from Schlotz et al. (2011) poor sleepers reported greater perceived stress reactivity levels, even after adjustment for age and BMI. However, we found that this association was not independent of chronic stress, which became the only significant predictor after being added into the regression model.

Accordingly, correlation analyses showed that chronic stress was more strongly associated with perceived stress reactivity than with sleep. In theory, chronic stress could have been a better predictor of perceived stress reactivity if reports of stress had been high while those of disturbed sleep had been modest. Yet, this was not the case as on average participants reported a relatively low mean score on the chronic stress scale (M = 1.1, SD = 0.1). Consequently, we assume that it is very likely that chronic stress impacts sleep quality as well as perceived stress reactivity and that this might lead to an artificial association between the latter two.

Our findings show furthermore that the sleep quality groups had dissociated response patterns with regard to their cortisol responses to the stress task and their generally perceived stress reactivity. This result is not unusual, as a recent review of the literature found the relationship between psychological and acute physiological stress (including cortisol responses) tends to be modest at best (Campbell & Ehlert, 2012). In our study perceived stress reactivity and acute cortisol reactivity were not only unrelated but they also showed a reversed pattern. This suggests that even though individuals have blunted cortisol responses in a stress task, the stress reactions they subjectively experience in their daily lives must not be lower than in persons who show higher cortisol responses. Consequently, studies which find blunted cortisol reactions cannot automatically infer that participants also perceive less stress when being confronted with a stressor. However, considering that the perceived stress reactivity scores used in this study were only based on three of the original six subscales, these findings are preliminary and need more empirical support. It would therefore be very beneficial if studies examining physiological stress responses also measured and reported perceived stress reactivity values or similar psychological measures. It must also be acknowledged that the Trier Social Stress Test for Groups is an artificial, standardized stressor which lasts for 14 min while the Perceived Stress Scale asks for stressful real life situations which vary in length. The stress response system does not react to all stressors in the same way and short-term stressors may mainly cause autonomic arousal with no or little involvement of the HPA-axis. Thus, our findings on the relationship

between subjective stress reactivity and the acute endocrine stress response must be interpreted with caution.

Limitations and Strengths

Our results need to be reported in light of their limitations. Sleep quality was measured with the Pittsburgh Sleep Quality Index (Buysse et al., 1989), the most comprehensive and widely used sleep questionnaire, but self-reported sleep ratings are prone to mood and memory biases and may also be influenced by psychosocial characteristics (Jackowska, Dockray, Hendrickx, & Steptoe, 2011). Indeed, sleep diaries (e.g., Monk et al., 1994) can provide a more precise estimate of sleep duration and quality than sleep questionnaires that are completed only once, but because sleep was not the main focus of the larger RCT this article is based on, we do not have this information. Objective and subjective sleep measures are only modestly correlated (Lauderdale, Knutson, Yan, Liu, & Rathouz, 2008), so it cannot be concluded that our findings would necessary be replicated if sleep was indexed objectively, as shown in Wright et al. (2007) where only actigraphy-based but not subjective sleep was associated with cortisol reactivity. Furthermore, males in our study were on average good sleepers, and when we tried to compute good and poor sleep groups using the recognised cut-off point of > 5 this resulted in a very small number of participants in the poor sleep category (n = 28), wherefore a median split was used instead. Unfortunately, this somewhat limits our conclusions and the comparability of our findings as previous studies used > 5 as cut-off point to split samples into good and poor sleep groups. Apart from that, our paper focused on associations with chronic stress but our data indicated that trait anxiety might also play a role with regard to perceived stress reactivity and sleep quality. It is also important to emphasize that potential participants were not specifically screened for sleep disorders such as sleep apnoea or insomnia. Lastly, the generalizability of our findings is constrained by our selective sample of healthy, middle-aged white men. In particular, we predominantly recruited sedentary men to this study, so it is unclear if factors such as BMI would be as strongly associated with sleep quality in the general population or among individuals who are more physically active.

Notwithstanding several limitations our findings advance the existing literature due to several strong study features: First, we assessed cortisol reactivity as an indicator for the reaction of the hypothalamic-pituitary-adrenal axis in response to the Trier Social Stress Test for Groups, which is a standardized and valid protocol to induce laboratory stress. Second, this study is, to the best of our knowledge, the first in this field to assess and investigate several known confounding variables and to use a large sample of working men, which helps to better integrate and understand existing findings on the examined topic. Thirdly, the study enables us for the first time to relate sleep quality and the endocrine stress response to the actual perceived stress response; this makes it possible to interpret physiological results in a more informed manner.

In conclusion, our study does not support past findings that individuals with lower sleep quality show increased cortisol responses to acute stress, when compared with those reporting good sleep. Previously reported associations between poor sleep quality and cortisol responses to acute stress may have potentially resulted from insufficient adjustment for covariates, in particular BMI. Thus, future studies relating sleep measures with stress reactivity responses should more carefully select covariates and consider the impact of chronic stress given its close ties with sleep patterns and the stress system. Finally, our study demonstrates that cortisol responses to acute laboratory stress and perceived general stress reactivity in daily life may have opposite patterns, as poor sleepers showed blunted cortisol responses while reporting greater subjectively perceived stress reactivity levels in general.

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Table 1

Characteristics of study participants

Variables	**Good sleep[1] Mean *(SD)***	**Poor sleep[2] Mean *(SD)***	***p***	***r***
Age	46.7 (10.2)	44.7 (10.0)	.30	
BMI	26.3 (3.2)	27.6 (3.6)	.07	
Trait anxiety	1.7 (0.4)	2.1 (0.5)	**<.001**	0.57
Physical exercise (min/week)[3]	33.0 (87.4)	19.8 (37.3)	.25	
Sleep quality overall[4]	2.9 (1.1)	7.2 (2.5)	**<.001**	0.84
Chronic stress level	1.0 (0.5)	1.5 (0.6)	**<.001**	0.41
Baseline cortisol level	10.3 (7.5)	8.9 (5.6)	.28	
Perceived stress reactivity	1.8 (0.4)	2.0 (0.5)	**.002**	0.28

Note. [1]Computed by median split (overall Pittsburgh Sleep Quality Index score ≤ 4); [2]computed by median split (overall Pittsburgh Sleep Quality Index score > 4); [3]Mean values are low and *SD* values are rather high as mainly sedentary participants have been recruited and 89 participants did not exercise at all; [4]Higher values indicate lower sleep quality; *SD* = standard deviation; BMI = body mass index; significant p-values are printed in bold.

Table 2

Bivariate correlations between study variables

Variables	BMI	Trait anxiety	Chronic stress	Sleep quality overall[1]	Sleep quality[2]	Cortisol reactivity	Overall cortisol response
Trait anxiety	**.22***	-					
Chronic stress	**.24****	**.78****	-				
Sleep quality overall[1]	.10	**.45****	**.44****	-			
Sleep quality[2]	.17	**.49****	**.41****	**.78****	-		
Cortisol reactivity	-.17	.02	-.09	-.07	-.13	-	
Overall cortisol response	**-.24****	-.08	-.16	-.15	**-.18***	**.91****	-
Perceived stress reactivity	.10	**.74****	**.64****	**.35****	**.28****	-.03	-.06

Note. [1]Higher values indicate lower sleep quality; [2]Dichotomous variable (0 = good sleep; 1 = poor sleep; correlation coefficients depict point-biserial correlation); BMI = body mass index; significant correlations are printed in bold, * = significant at the .05 level, ** = significant at the .01 level.

Table 3

Associations between sleep quality, cortisol reactivity and perceived stress reactivity

	Cortisol reactivity sample 1-3			**Overall cortisol response**			**Perceived stress reactivity**		
	b	95% CI	*p*	*b*	95% CI	*p*	*b*	95% CI	*p*
Model 1									
Sleep quality[1]	-4.623	(-11.28 to 2.04)	.172	-18.246	(-35.98 to -0.51)	**.044**	0.253	(0.10 to 0.41)	**.002**
Model 2									
Sleep quality[1]	-3.778	(-10.59 to 3.03)	.274	-13.086	(-30.90 to 4.73)	.148	0.235	(0.07 to 0.40)	**.005**
Age	-0.040	(-0.37 to 0.29)	.806	0.483	(-0.37 to 1.34)	.264	-0.003	(-0.01 to 0.00)	.386
BMI	-0.764	(-1.75 to 0.22)	.127	-3.442	(-6.01 to -0.87)	**.009**	0.009	(-0.01 to 0.03)	.439
Model 3									
Sleep quality[1]	-3.525	(-10.92 to 3.86)	.347	-11.051	(-30.36 to 8.26)	.259	0.018	(-0.12 to 0.16)	.797
Age	-0.042	(-0.37 to 0.29)	.798	0.466	(-0.39 to 1.32)	.283	-0.002	(-0.01 to 0.01)	.615
BMI	-0.745	(-1.75 to 0.26)	.146	-3.294	(-5.93 to -0.66)	**.015**	-0.007	(-0.03 to 0.01)	.492
Chronic stress	-0.547	(-6.61 to 5.52)	.859	-4.409	(-20.26 to 11.45)	.583	0.470	(0.36 to 0.59)	**<.001**

Note. [1]Dichotomous dummy variable (0 = good sleep; 1 = poor sleep); BMI = body mass index. Results are presented as unstandardized regression coefficients (*b*), 95% confidence intervals (CI) and p-values; significant p-values are printed in bold

Figures

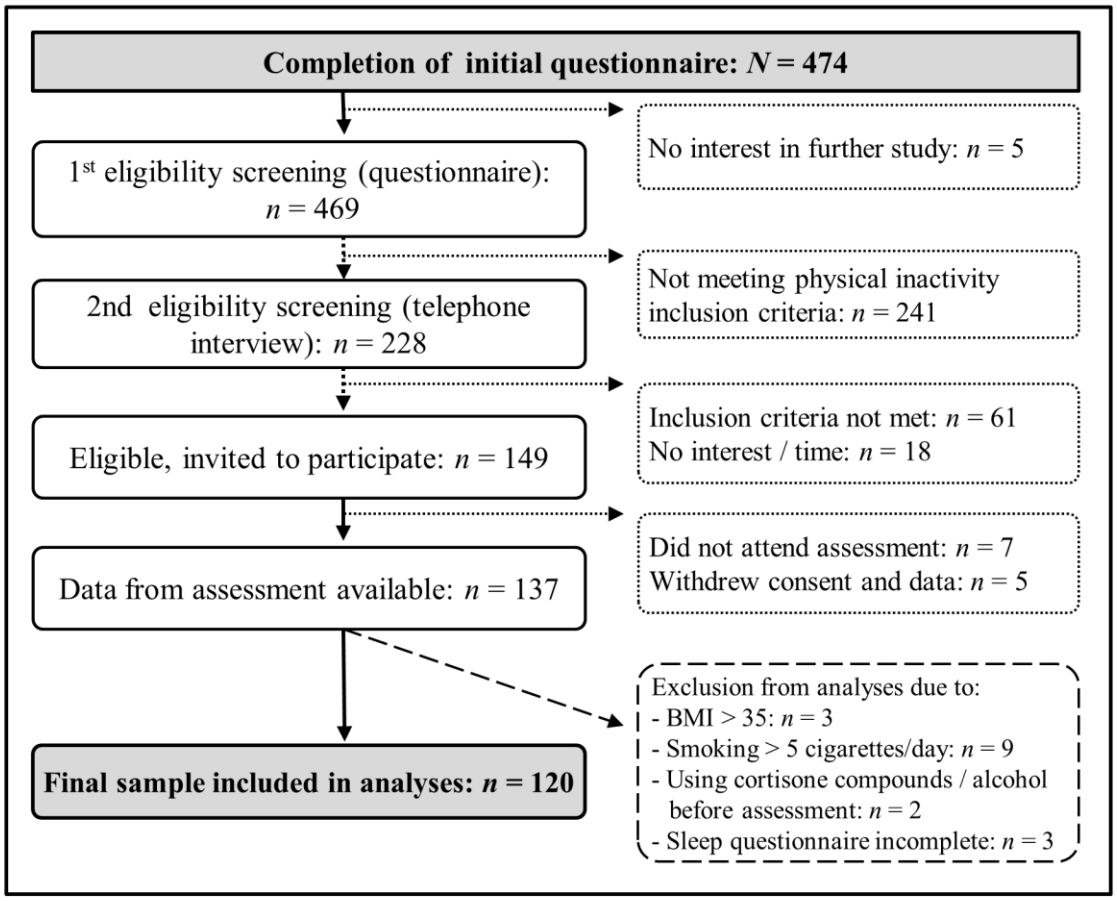

Figure 1. Detailed participant flow chart

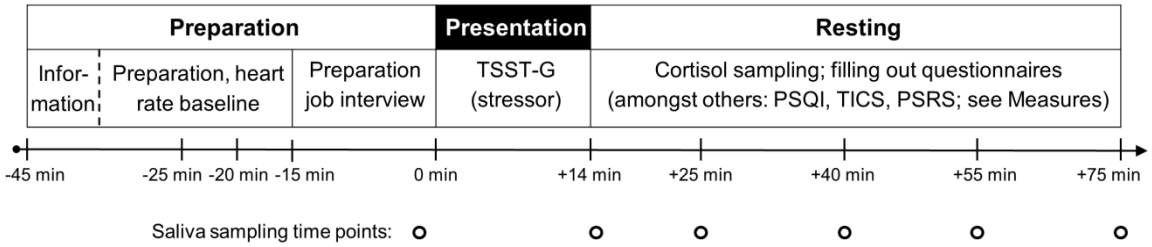

Figure 2. Sequence of events and measurements during the stress response assessment (TSST-G = Trier Social Stress Test for Groups; PSQI = Pittsburgh Sleep Quality Index; TICS = Trier Inventory for Chronic Stress; PSRS = Perceived Stress Reactivity Scale).

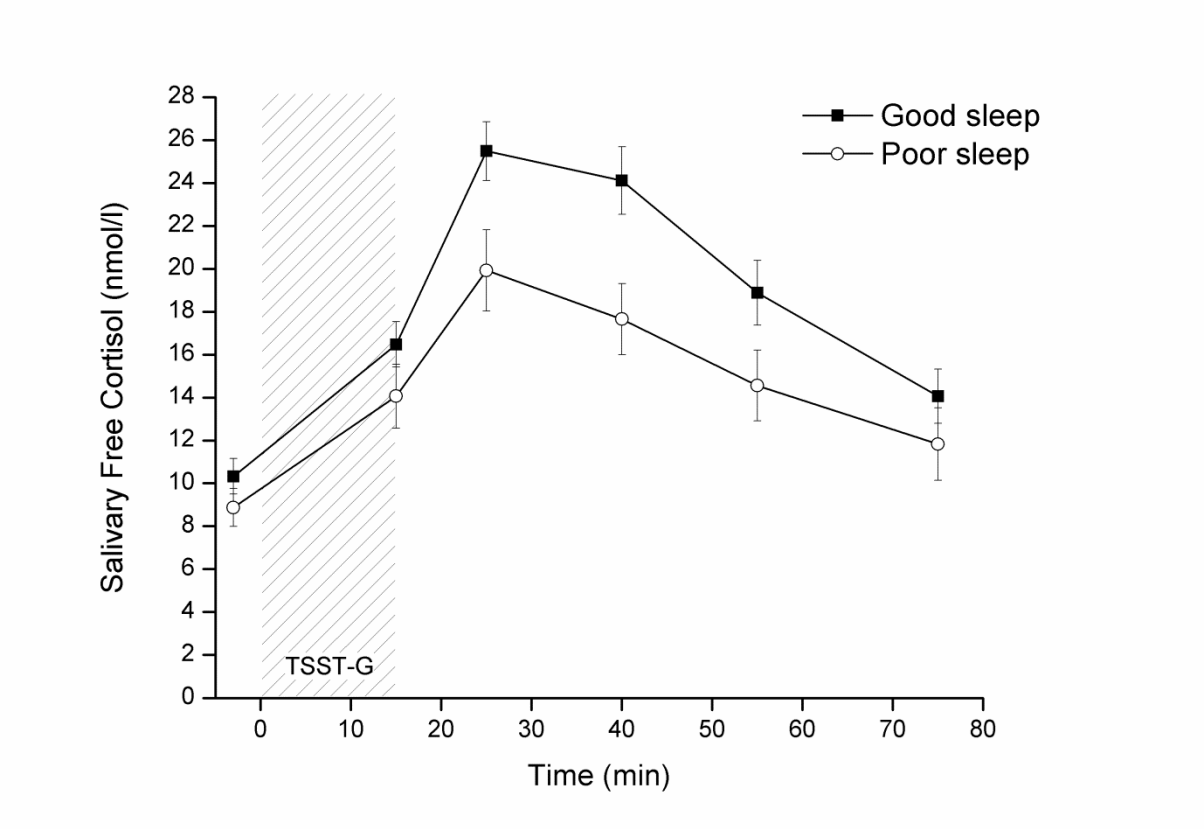

Figure 3. Mean salivary free cortisol levels (untransformed values) before, during, and after the psychosocial stressor (Trier Social Stress Test for Groups = TSST-G, shaded area) in the groups with good and poor sleep. Error bars are SEM.